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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

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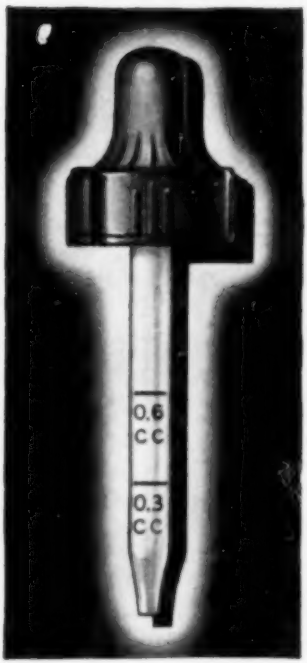

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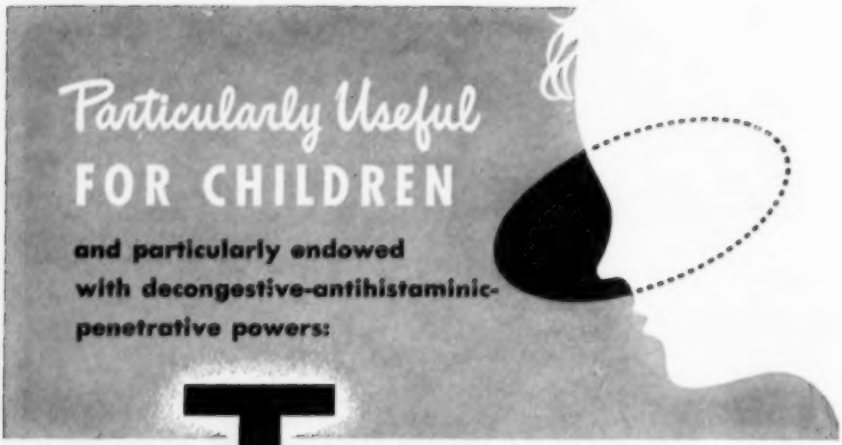

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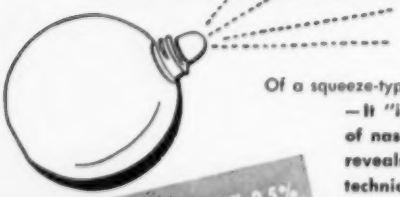

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1. Silbert, N. E.: *GP*, 8:35, Dec., 1953.

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2. Helmer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951.
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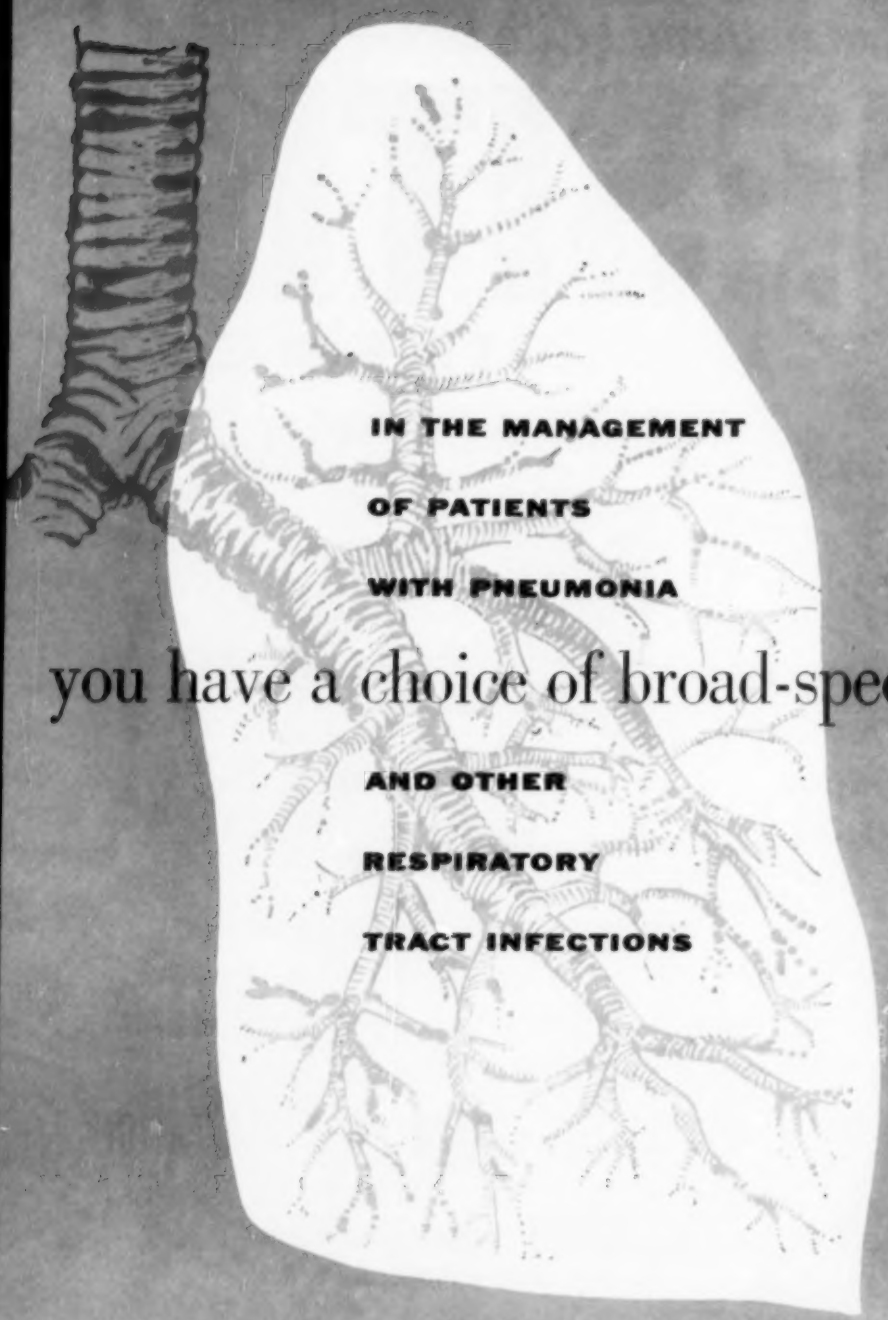

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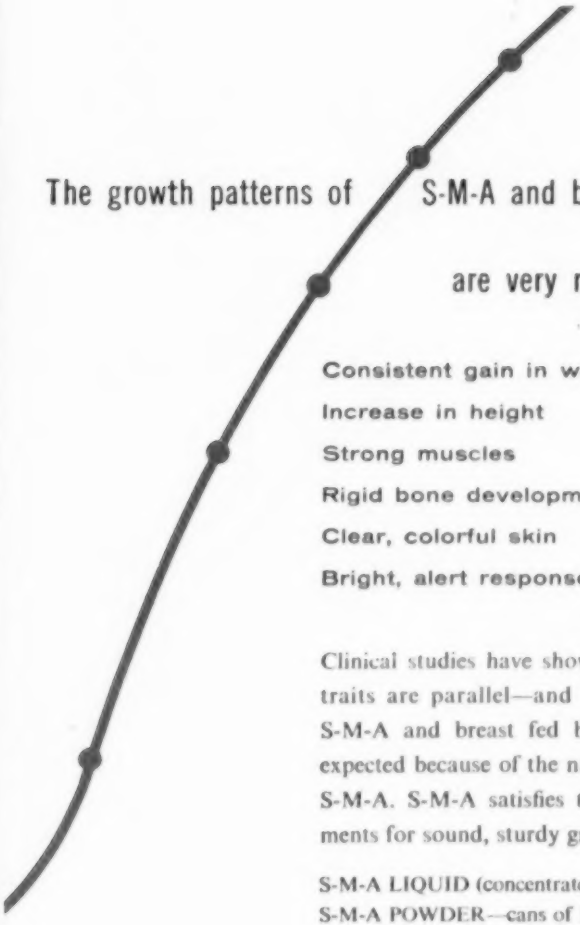

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2. Curry, J. C. and Barber, F. W.: Bacteriological Proceedings, 1951, of The Society of Am. Bact., page 23.
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RECURRING ABDOMINAL PAIN IN CHILDREN SECONDARY TO SINUSITIS

EDWARD E. BROWN, M.D., F.A.A.P.
Ashland, Ore.

While most cases of recurring abdominal pain in childhood are said to be of undetermined origin,[1-5] in my experience with several hundred such complaints the most frequent incitant has been a chronic sinusitis (Figs. 1-4). This evidence is presented, the pertinent literature is reviewed, and the conclusions of some authors are reevaluated.

The site of pain is most often in the umbilical area (Fig. 1), with or without involvement of the right lower quadrant or other areas.[2-4] Ages of affected children were two to eleven years, most often three to seven years, as noted also by Conway.[3]

ETIOLOGY

Recurring pain in the abdomen has predisposing and immediate causes. For example, abdominal pain which follows an upper respiratory infection (the predisposing cause) may actually be due to gas, or to inflammation of the intestine, appendix or mesenteric nodes (the immediate cause).

Predisposing causes are mainly upper respiratory infections and allergy, while *immediate causes* are the abdominal diseases which are attended by sudden distention of the bowel, intestinal inflammation, obstruction or hemorrhage, and the thoracic diseases which refer pain to the abdomen.

In approximate order of frequency, the diseases accounting for

abdominal pain in children are: colic (distention by gas), mesenteric adenitis, enteritis, appendicitis, subacute rheumatic fever, Henoch's purpura, pyelonephritis, and reflex pain from right lower lobe pneumonia and pleurisy. Abdominal pain exists in cyclic vomiting, but it is not a prominent symptom.[6] In the production of gas pain or intestinal spasm, allergy is frequently incriminated,[2, 7-10] although some observers find this cause but rarely.[3, 4] I have recognized allergy as a not infrequent cause of colic in infancy, but much less often in children.

FIG. 1. Acute abdominal pain associated with active sinusitis. Child had just been brought home from school by principal. History of head cold two weeks ago and occurrence of cold yesterday. Child had frequent colds, occasional headaches, and mild cervical adenitis. Note marked puffs beside nose and photophobia due to chronic pansinusitis.

Conway's[3] admirable and controlled study is worthy of elaboration. Among 243 children with abdominal pain, the final diagnosis was as follows: colic of unknown cause (75 cases); mesenteric adenitis (52 cases); periodic syndrome (cyclic vomiting) (24 cases); constipation (28 cases); psychogenic (23 cases); enteritis,

food allergy, indigestion, over-eating, air swallowing and threadworms (23 cases); cystitis or pyelitis (3 cases); pleural effusion (2 cases); acute appendicitis (2 cases), and other unusual causes.

Judging from my own observations, an overlooked sinusitis may have existed in two of the above groups, namely, colic of unknown cause, and the so-called psychogenic group. Small children with colic frequently reveal evidence of an associated upper respiratory infection at the time, such as "snuffly breathing,"[5] whereas in the psychogenic group, Conway states that "nausea is the commonest symptom" and that "many have an accompanying head cold." These two symptoms, nausea and head colds, clearly indicate the possibility of sinusitis.[11] As is true with most investigators of the causes of abdominal pain, Conway does not discuss sinusitis, nor is it considered in the above classification. Failure to diagnose sinusitis is the most glaring oversight among the infections of children.[12]

Evidence for chronic sinusitis as a frequent cause of abdominal pain is discussed under the following headings: (1) seasonal variations; (2) presence of colds and other symptoms of sinusitis; (3) diseases associated with both abdominal pain and sinusitis; and (4) response to treatment of sinusitis.

1. *Seasonal Variations.* Comparatively few cases of abdominal pain in children are encountered in hot summer weather. However, in the fall, abdominal pain becomes a prominent symptom, complaints being frequent also in the winter and spring. Abdominal pain, when encountered in summer weather, is often associated with gastroenteritis and allergy.

2. *Presence of Colds and other Symptoms of Sinusitis.* I have found sinusitis usually present in children with recurring abdominal pain, and conversely, children with chronic sinusitis often complain of abdominal pain. McKenzie[13] finds that many "patients with chronic sinusitis will have gastrointestinal symptoms or complications, nausea, vomiting, diarrhea, fever and pain, either general or localized in the region of the appendix or gall bladder." Sinusitis may be considered as only a "cold,"[2] whereas the frequently recurring cold is almost invariably an acute exacerbation of chronic sinusitis.[11, 14]

The most common symptoms observed in children with recurring abdominal plain are anorexia, particularly for breakfast, nausea, vomiting, head cold, cough, low-grade fever, fetor oris,

photophobia, headache and pain in the legs (Fig. 2). These symptoms were prominent in the study of Conway and others.[13, 16] These are all symptoms of chronic sinusitis.[11] Although other causes exist for each symptom, only sinusitis produces all of them.[11]

Signs of sinusitis[16] frequently present in children with abdominal pain include circles and puffs under eyes, puffs beside nose (Figs. 1-4), nasal obstruction, mouth-breathing (Figs. 2-4), photophobia

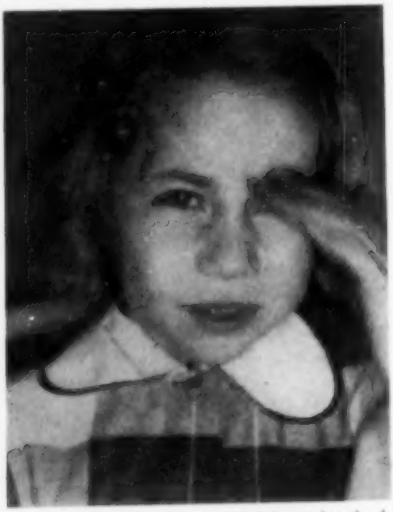

FIG. 2. Girl, aged 6, complaining of pain in abdomen and knees. Child has chronic pansinusitis, almost complete nasal obstruction, and moderate cervical adenitis. Note broad base of nose, mouth-breathing and photophobia, all caused by sinusitis.

(Figs. 1, 2, 4), sinus tenderness, and cervical adenitis[17] despite a previous tonsillectomy in many of these children.[3] The cervical adenitis is secondary to bacterial toxins from sinusitis, and the mesenteric adenitis may have a similar origin (*infra*).

3. *Diseases Associated with both Abdominal Pain and Sinusitis.* Among the diseases associated with abdominal pain, chronic sinusitis is especially concerned with mesenteric adenitis, appendicitis, "primary" peritonitis, rheumatic fever, Henoch's purpura and some cases of so-called colic. These diseases are discussed.

Colic. This is an agonizing and intermittent pain. It comes on suddenly and usually ends as abruptly. There is very little abdominal tenderness and pressure is often a source of relief.[1] Various explanations are given for its occurrence—allergy, constipation, laxatives and oxyuris.[18] An acute rhinitis or sinusitis commonly produces colic in an infant, perhaps in part due to distention by gas and to inflammation of the intestinal tract.

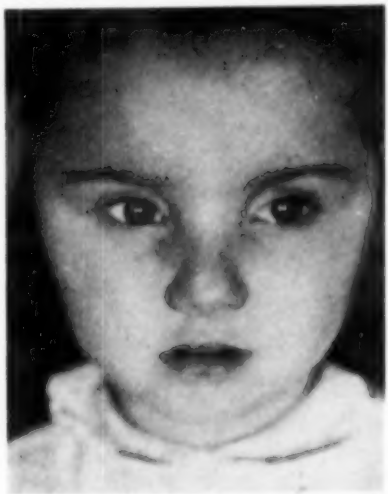

FIG. 3. Abdominal pain associated with sinusitis and hypertrophied, injected tonsils. Note puffs beside nose and mouth-breathing.

Mesenteric Adenitis. Nonspecific mesenteric lymphadenitis is of unknown etiology[19-21] and may occur at any time during an upper respiratory infection.[1, 3, 21-25] In Ireland's series, 68 per cent of 22 patients showed evidence of infection of the upper respiratory tract at the time of operation or just preceding it, and a higher percentage might have been found if such evidence had been especially sought.[24] Baker and James[21] found pharyngitis uniformly present, while Goldberg[22] noted that the throat is usually red, and there was frequently a great deal of nasal discharge. These are common symptoms in chronic sinusitis. Since the mesenteric

nodes are sterile,[20, 24] I believe that the pathogenic agent is toxin, rather than bacteria, either hematogenous or absorbed from the intestinal tract. The toxin is produced mainly by streptococci,[26] associated with "colds"[14] or flare-ups of chronic sinusitis. Fitzsimons[27] believes the causative agent may gain access to the lymphatics by way of the terminal ileum, since he found this portion of the bowel often definitely injected, thickened and edematous. Wilensky[26] is probably correct in stating that both the throat and

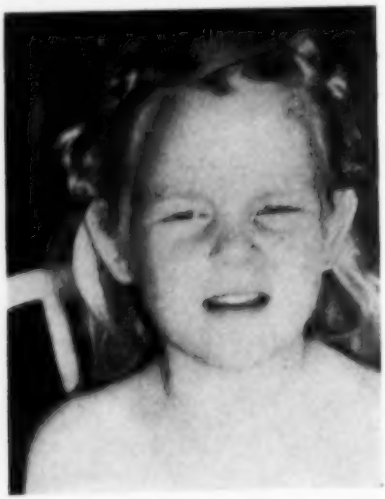

FIG. 4. Pneumonia (second day) and abdominal pain, secondary to sinusitis. Note marked puffs beside nose, photophobia and mouth-breathing.

the terminal ileum are the most common ports of entry of the infection. The involvement of the cervical nodes parallels that of the mesenteric nodes, occurring mainly in children usually between ages 2 and 13.[17, 20, 21]

Abdominal tenderness may be diffuse or have a predilection for the right side[21, 27] Muscle spasm is absent. Fever is usually present[21], but may be absent.[17] Mesenteric adenitis is often diagnosed during surgery, after confusion with acute appendicitis.[23, 24] Ger[17]

recently reported his ability to palpate mesenteric lymph nodes in one-fourth of the cases under his observation.

Acute and Subacute Appendicitis. Even in this more serious cause of abdominal pain, acute sinusitis[29, 30] and the upper respiratory infections are frequent percursors.[31-34] Brennemann[1] states that "there is considerable evidence that more than half of all cases of appendicitis are causally related to throat infections," while Evans[34] finds upper respiratory tract infection in 86 per cent of cases of "epidemic" appendicitis, either at the onset or at an average of eight days earlier. Enlarged cervical nodes are usually present during an attack of appendicitis.[35] The infecting organism in young children is usually the streptococcus.[36] Dorsey[30] demonstrated the similarity (culturally and morphologically) of streptococci obtained from the appendix and nasopharynx of the same patient.

"Primary" Peritonitis. This rare producer of abdominal pain may be incited by acute sinusitis.[21] A preceding respiratory infection is noted in from 70 to 100 per cent of cases.[37-40]

Leopold and Kaufman[39] believe that the nasopharynx is the most common portal of entry. They state that, while a few authors believe that the organisms are swallowed and then migrate through the wall of the intestine, especially in the ileocecal region, most authors believe that bacteria reach the peritoneum from the nasopharynx through the blood stream.

Rheumatic Fever. Before joints are involved in rheumatic fever, abdominal pain may precede by two or more days.[2, 41] Abdominal pain is one of several prerheumatic symptoms.[42, 43] Among 162 rheumatic children, Coburn[44] observed acute abdominal pain in 32, often at the onset of a recrudescence. Abdominal tenderness may be considerable. The cause of abdominal pain in rheumatic fever is uncertain; suggested causes include peritonitis,[8] lymphadenitis[45] or involvement of abdominal muscles.[45]

Chronic sinusitis exists in 100 per cent of rheumatic children[46] and is the probable cause of rheumatic fever[47] and of the frequently associated mesenteric adenitis, purpuric lesions and so-called "rheumatic appendicitis."

Henoch's Purpura. This uncommon cause of abdominal pain[8, 9] is probably due to streptococcic toxins whose source is the upper respiratory tract. Hemolytic streptococci are often found in the nasopharynx.[48, 49] Capillary fragility[28, 50] and purpuric lesions prob-

ably result from streptococcal toxins[20, 51, 52] acting directly on the vessels. Coburn[53] reported scattered purpuric lesions in the peritoneal cavity and other areas in rheumatic children.

4. *Response to Treatment of Sinusitis.* Before intelligent treatment of abdominal pain can be instituted, the cause must be determined. One must rule out surgical and other serious conditions which exist in about 5 per cent of children complaining of abdominal pain.[2]

The large group of cases secondary to upper respiratory infections responds well to treatment of an associated active sinusitis. The nose is kept open with isotonic, astringent nose drops or the newer sprays (e. g. Neo-cortef nasal spray; Vasocort Spraypak), followed by hot wet towels to the face, after applying cold cream. This routine is carried out three times daily, more often if the nose is obstructed. In younger children, steam inhalations may be substituted for hot packs. The head-up posture[54] is used during sleep, and Fowler's sitting-up posture is maintained during the waking hours. Chilling, fatigue and, when possible, allergens must be avoided. A penicillin injection should be added to the above treatment when abdominal pain is secondary to acute sinusitis; relief is often obtained within a day.

To prevent recurrences of abdominal pain in children, treatment of chronic sinusitis twice daily should be pursued indefinitely.

Even mild cases of appendicitis, secondary to sinusitis, pharyngitis and tonsillitis are aided by the above treatment. The favorable response of cases of subacute appendicitis to penicillin[55] may be due in part to bacteriostasis of streptococci located in the sinuses and other foci as well as in the infected appendix. Pain and nausea in many cases disappear in a day or two.

DISCUSSION

Causes of abdominal pain receiving prominence in the literature include upper respiratory infections,[2, 18] tonsillitis[2, 4] (Fig. 3), ear infections,[4] pneumonia[1, 4, 8, 9, 28, 56] (Fig. 4) particularly in the right lower lobe,[1, 57] diaphragmatic pleurisy,[1, 9, 28, 56] mesenteric adenitis, rheumatic fever, Henoch's or allergic purpura, appendicitis and "primary" peritonitis.

The most consistent focus in all these infections is chronic

sinusitis.[12, 46] The striking feature which makes chronic sinusitis more important than other foci in children is the development of pus under tension. Confined pus is a requisite for a true focus of infection.[13] Cervical adenitis, often present in children with abdominal pain, is consistently present when sinusitis is causative. Pus is never confined in pharyngitis, adenoiditis and rhinitis. It may be confined in mastoiditis and alveolar abscess, and to a lesser extent in otitis and tonsillitis.

Toxins tightly confined in foci penetrate surrounding mucous membranes and other enveloping tissues to enter the lymphatics and blood stream, thus lowering capillary resistance,[57, 58] producing petechiae[57] and purpuric hemorrhages, invading the appendix, mesenteric nodes and peritoneum. By some of these mechanisms one may explain the vagaries of streptococcal toxin in producing recurring abdominal pain.

CONCLUSIONS

1. Chronic sinusitis and associated upper respiratory infections are the most common predisposing causes of recurring abdominal pain in children.

2. The complaint of abdominal pain is encountered usually during an acute exacerbation of a chronic sinusitis, often labeled a cold or an upper respiratory infection. A glance at the child will often reveal many signs of sinusitis, notably puffs beside nose, circles under the eyes, mouth-breathing and photophobia. History and physical examination will disclose many other symptoms and signs.

3. Recurring abdominal pain is a symptom in diseases often secondary to chronic sinusitis, such as mesenteric adenitis, appendicitis, rheumatic fever, Henoch's purpura and "primary" peritonitis.

4. Daily treatment of chronic sinusitis, as outlined, prevents many recurrences of abdominal pain. For abdominal pain secondary to either an acute sinusitis, or to an acute flare-up of a chronic sinusitis, parenteral penicillin should be included in treatment.

5. One must never lose sight of surgical and other serious causes of abdominal pain, existing in about 5 per cent of cases.

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SUBCLINICAL FORMS OF TUBERCULOUS MENINGITIS. (Minerva pediat., 6:265-275, April 30, 1954). Gentili and Paci state that early diagnosis of tuberculous meningitis is difficult because the disease frequently differs from the classical picture in its course. Tuberculous meningitis may have an intermittent course during which periods of violent symptoms alternate with periods of remission that simulate a complete recovery. The condition may be subacute with a slow onset and symptoms suggesting a cerebral tumor. There are also oligosymptomatic forms and forms with an atypical course. Among the last named is meningitis that heals spontaneously and allergic tuberculous meningitis. Early diagnosis of tuberculous meningitis can be made only through studies of the spinal fluid. A greater number of early diagnoses can be obtained by performing a spinal puncture in patients who are known to have tuberculosis and in whom there is evidence that the process might extend. This "control" spinal puncture in these patients is the best means of diagnosing inflammation of the menges before the clinical signs become apparent. Early and adequate therapy can thus be instituted, and this may prevent the onset of the clinical signs and bring about recovery before the nervous system becomes involved.—*J.A.M.A.*

PENICILLIN TREATMENT FOR EARLY CONGENITAL SYPHILIS*

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In an attempt to determine an acceptable schedule of penicillin treatment for early congenital syphilis, or at least to establish limits of treatment adequacy, the Venereal Disease Program began in 1946 to collect case histories for analysis from a number of its treating agencies. Records on patients treated during the period 1946-1950 were submitted by treatment centers located at St. Louis; Louisville; Norfolk; Hot Springs; Charlotte, N. C.; and Charleston, W. Va. (Table 1). Certain clinics originally

Table 1. Clinics Included in the Study by Number of Patients Treated and Percentage of Patients Following 18-21 Months

Clinic	Number Treated	Percent Observed 18-21 Months
St. Louis	91	54
Charlotte, N.C.	53	38
Hot Springs	189	30
Norfolk	9	33
Charleston, W. Va.	59	48
Louisville	71	34
Total	472	38

participating in the study were omitted from the evaluation because of their inability to obtain posttreatment information on a sufficient number of patients.

THE STUDY GROUP

The evaluation presented is based on 472 patients having early congenital syphilis. Criteria for inclusion in the study were as

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follows: (a) Children under 3 months of age at time of treatment were included only if there was physical evidence of congenital syphilis, such as snuffles, a cutaneous syphilis, or bone malformation; (b) Children 3-24 months of age at time of treatment were included on the basis of positive serologic tests for syphilis with or without the presence of clinical manifestations. The exclusion from the study of children under 3 months of age at time of treatment, whose only reported evidence of syphilis was a positive blood test, was based on the fact that the positivity might possibly have been caused by transfer of the mother's reagin. All study patients were treated with penicillin, with or without adjunctive bismuth, during the first 2 years of life.

Approximately 40 per cent of the 472 patients included in the study were observed for a period of at least 18-21 months, and were used as a basis for determining the status of the study group according to a modified life table technique[7]. Experience in therapy determination in early syphilis has shown that the outcome at 18-21 months is essentially no different from that at 24 months, except for the increased possibility of reinfection. With reinfection presenting no problem in this study group, it is felt that the data here presented give a valid indication of the effectiveness of penicillin in the production of seronegativity and in the prevention of clinical and serorelapse during the first 2 years following treatment for early congenital syphilis. This period of observation is, of course, far too limited to provide any information regarding the adequacy of penicillin treatment in preventing the development of late clinical manifestations of congenital syphilis.

METHOD OF ANALYSIS AND RESULTS OF THE STUDY

Interpretation of the collected data was, in general, the same as in use in the therapy evaluation for acquired syphilis[8]. Interpolation of dates of reversal of serology from positive to negative was necessary in many cases. Therapy has been evaluated on the basis of (a) clinical or serologic failure necessitating retreatment, and (b) the posttreatment serologic behavior according to (1) the age of the patient, (2) the total dosage of penicillin, and (3) the amount of penicillin per kilogram of body weight.

By the 18-21 month period following treatment, 10 patients among those observed had been classified as clinical or serologic

Table 2. Summary of History and Treatment Data on Patients 2 Years of Age or Younger Classified as Failures After Treatment for Congenital Syphilis

Patient Number	Penicillin Administered: Total Amount	Penicillin Administered: Units Per Kilogram Weight	Penicillin Administered: Type	Age at Time of Treatment	Darkfield Status at Treatment	Serologic Test for Syphilis at Treatment: Type	Serologic Test for Syphilis at Treatment: Results (in Kahn units)	Treatment Failure: Type	Treatment Failure: Date (in relation to date of Treatment)
1	150,000	43,000	Aqueous	1 month	Lesion present, darkfield positive	No Data	No data	Treatment death	Less than 30 days
2	300,000	81,000	Aqueous	2 months	Lesion present, darkfield positive	Kahn	1360	Clinician's decision	30-59 days
3	300,000	No Data	Aqueous	2 months	Lesion present, darkfield positive	No Data	No Data	Clinical relapse	4-5 months
4	630,000	252,000	Aqueous	2 months	Other clinical evidence	Kahn	256	Clinician's decision	8-9 months
5	675,000	130,000	Aqueous	4 months	Lesion present, darkfield positive	Kahn	256	Sero-relapse	5-6 months
6	675,000	199,000	POB	1 month	Lesion present, darkfield positive	Kahn	512	Sero-resistance	6-7 months
7	1,200,000	120,000	Aqueous	20 months	No clinical evidence	Kahn	64	Sero-resistance	12-15 months
8	1,800,000	281,000	POB	20 months	No clinical evidence	Kahn	512	Clinician's decision	12-15 months
9	2,700,000	270,000	POB	18 months	No clinical evidence	Kahn	512	Clinician's decision	6-7 months
10	3,000,000	462,000	POB	7 months	Lesion present, darkfield positive	Kahn	2048	Clinical relapse	3-4 months

failures. Of these 10 patients, one child died during treatment. Of the remaining nine patients, there were four re-treated on the basis of what was called "clinician's decision." Review of these patients showed no evidence of clinical failure. All had shown significant to progressively sustained fall in serologic titer, in one to negativity, which, in light of today's knowledge of variation in speed of serologic reversal following treatment, would be considered as satisfactory. However, at the time these patients were being observed, and under clinical conditions then prevailing, the serologic variability was not so well understood and the tendency existed to re-treat when in doubt so as to give the patient the maximum of potential for cure. Two patients were re-treated for seroresistance, two patients were re-treated for clinical relapse, and one was re-treated for serorelapse. After

Table 3. Status of Study Patients at 18-21 Months Following Treatment by Age at Time of Treatment

Age at Time of Treatment	Number Treated	Number Observed	Percentage Seronegative	Percentage Seropositive	Percentage of Failure Serologic	Clinical
Under 3 months	107	38	92.1	-	1.7	6.2
3-5 months	139	52	95.1	3.8	1.1	-
6-11 months	96	44	80.7	17.9	-	1.4
12-24 months	130	47	42.4	52.6	5.0	-

adjustment for patients lost from observation, this represents a re-treatment rate of 3.5 per cent for the total study group. This composite rate is more meaningful when it is expressed specifically in relation to age of patient and amount of penicillin administered and will be discussed later in this report.

A summary of the histories of those re-treated is presented in Table 2, with the patients arranged in order by total amount of penicillin administered. It is evident from this table that no particular combination of history and/or treatment factors appears consistently among those re-treated. It is true that the serologic titer at time of treatment among those for whom the information was available was, in every case, 64 units or more. However, this preponderance of high titers loses much of its significance in light of the fact that 85 per cent of the entire study group had titers of 64 Kahn units or higher at time of treatment.

Table 4. Status of Study Patients at 18-21 Months Following Treatment by Amount of Penicillin Administered

Amount of Penicillin	Number Treated	Number Observed	Percentage Seronegative	Percentage Seropositive	Percentage of Failure Serologic	Percentage of Failure Clinical
Less than 800,000	76	38	87.8	2.4	3.3	6.5
800,000 - 1,199,999	46	12	75.0	25.0	-	-
1,200,000 - 1,799,999	109	40	73.5	24.5	2.0	-
1,800,000 - 2,399,999	115	40	78.3	19.6	2.1	-
2,400,000 - 3,599,999	95	36	72.4	24.1	1.9	1.6
3,600,000 or more	29	14	64.3	35.7	-	-

Table 5. Status of Study Patients at 18-21 Months Following Treatment by Age by Total Amount of Penicillin

Age Group and Amount Penicillin	Number Treated	Number Observed	Percentage Seronegative	Percentage Seropositive	Percentage of Failure Serologic	Percentage of Failure Clinical
Under 1 Year of Age:						
Less than 2,400,000 units of penicillin	280	101	89.0	7.7	1.2	2.1
2,400,000 units of penicillin or more	61	32	91.8	6.1	-	2.1
1-2 years of age:						
Less than 2,400,000 units of penicillin	66	29	48.7	45.4	5.9	-
2,4000,000 units of penicillin or more	63	18	1/ 32.2	64.4	3.4	-

1/ This anomalous relationship between amount of penicillin and the attainment of seronegativity is explained by the fact that the larger amounts of penicillin are given to the older and heavier children in whom the attainment of seronegativity is slowest.

Table 6. Status of Study Patients at 18-21 Months Following Treatment by Units of Penicillin Per Kilogram of Body Weight, by Type of Penicillin

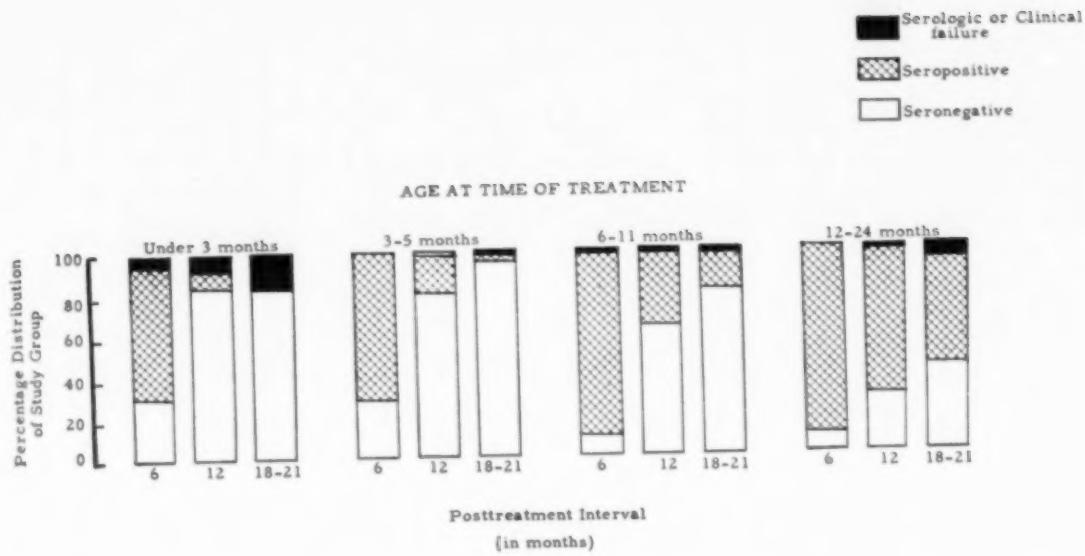
Units of Penicillin Per Kilogram of Body Weight	Number Treated	Number Observed	Percentage Seronegative	Percentage Seropositive	Percentage of Failure	
					Serologic	Clinical
Aqueous						
40,000 - 160,000	65	36	79.4	12.8	4.2	3.6
161,000 - 320,000	115	41	72.0	26.4	-	1.6
321,000 or more	64	28	85.7	14.3	-	-
PCB						
40,000 - 160,000	8	2	-	-	-	-
161,000 - 320,000	74	16	49.8	38.8	11.4	-
321,000 or more	40	15	95.5	-	-	4.5

The serologic and clinical outcome of penicillin therapy at 18-21 months posttreatment is shown in Table 3 by age of patient at time of treatment. The rate of serologic failure was 1.7 per cent among those under 3 months of age at treatment, as compared to 1.1 per cent for those 3-5 months of age, no serologic failures among those 6-11 months, and 5.0 per cent for those 12-24 months of age. A clinical failure rate of 6.2 per cent occurred among those under 3 months of age, and 1.4 per cent among those 6-11 months of age. There were no clinical failures among those 3-5 months and 12-24 months of age at time of treatment. In relation to serologic outcome, there is a direct correlation between the age at time of treatment and the per cent remaining positive after treatment. Among those patients under 3 months of age at time of treatment there were no seropositive results at 18-21 months posttreatment. Among those 3-5 months the rate of positivity was 3.8 per cent, and in the group 6-11 months, 17.9 per cent. Among those children treated during the second year of life, 52.6 per cent had positive serologic findings at 18-21 months after treatment. This finding merely serves to emphasize the fact that the longer the delay in treatment the slower the rate of seroreversal. In this respect, congenital syphilis responds serologically in much the same manner as acquired syphilis.

Posttreatment spinal fluid examinations were done on 56 (approximately 12 per cent) of the 472 patients treated. In this group no positive spinal fluid test results were reported. Among the 130 patients, 12-24 months of age at time of treatment, a group in which an unusually high rate of seropositivity persisted during the follow-up period, 12 had posttreatment spinal fluid examination; 7 of these patients had sustained positive blood tests since treatment, yet the spinal fluid test results were negative.

The status of study patients at 18-21 months following treatment is shown in Table 4 by total amount of penicillin administered. As the amount of penicillin is increased, the percentage becoming seronegative becomes smaller. This anomalous relationship between amount of penicillin and the attainment of seronegativity is perhaps explained if the optimum dosage in the treatment of congenital syphilis is determined in terms of units of penicillin in kilogram of body weight as shown in Table 6. This

Figure 1. Posttreatment Serologic and Clinical Status of Study Group, by Age at Time of Treatment, at Periodic Intervals After Treatment



would mean that the larger total amounts of penicillin approaching adult doses are given to the older and heavier children, in whom the attainment of seronegativity is slowest as shown by Table 5. Likewise, the distribution of serologic and clinical failures by amount of penicillin reflects the influence of age and weight in the selection of dosage schedule.

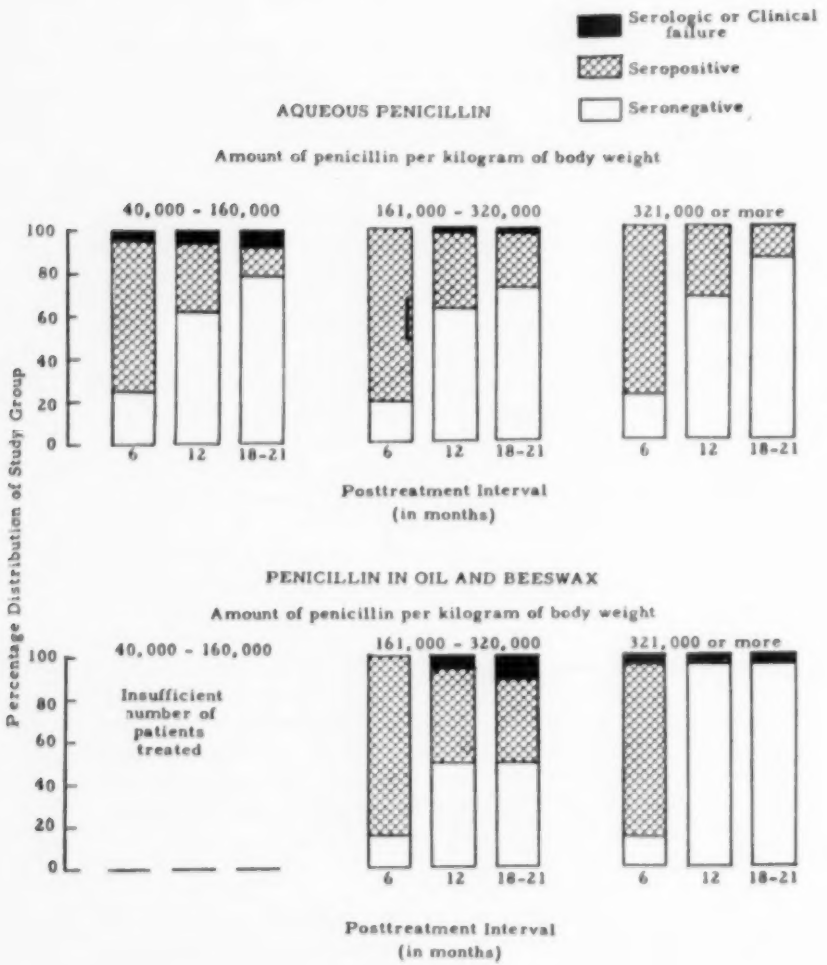
The importance with respect to seroreversal of the early diagnosis and treatment of congenital syphilis is demonstrated quite effectively by the results shown in Table 5. Regardless of the amount of penicillin administered, a significantly greater percentage of children treated during the first year of life attain negativity within 18-21 months after treatment than do children treated during the second year of life. This is an important factor because serofastness in the adequately treated congenitally syphilitic child can give rise to much disquiet for both child and family and often leads to needless stigmatization of the child as well as re-treatment. Figure 1 presents graphically the results obtained with varying amounts of penicillin by age intervals.

The status of patients at 18-21 months by amount of penicillin per kilogram of body weight is shown in Table 6. The highest percentage of seronegativity and the lowest percentage of serologic or clinical failure in both the group receiving aqueous penicillin and that receiving penicillin in oil and beeswax (POB) are found among those patients receiving 321,000 units or more of penicillin per kilogram of weight. The progressive status of the patients at 6 months, 12 months, and 18-21 months posttreatment by type of penicillin and amount administered per kilogram of weight is shown in Figure 2. It is apparent that best results were obtained among patients receiving 321,000 units or more of penicillin per kilogram. Of those receiving aqueous penicillin in this treatment category, 85.7 per cent had attained negativity, 14.3 per cent remained seropositive, and no serologic or clinical failures had occurred at 18-21 months posttreatment. Among those treated with POB, 95.5 per cent had attained negativity but the remaining 4.5 per cent had experienced clinical failure.

DISCUSSION

The results of penicillin therapy for congenital syphilis during the first 2 years of life are, in a manner of speaking, analogous

Figure 2. Posttreatment Serologic and Clinical Status of Study Group, by Units of Penicillin per Kilogram of Body Weight, at Periodic Intervals After Treatment



to treatment results in acquired syphilis during the early stages and latency. Since all of the patients in the study treated during the first 3 months of life had clinical symptoms of early congenital syphilis, they simulate patients having primary and secondary acquired syphilis, and the results of treatment in this group are quite similar to those found in treatment for early syphilis. After treatment with any reasonable amount of penicillin, the percentage of seronegativity attained in each group by the second year after treatment is quite high, and patients in need of further treatment will have relapsed or showed seroresistance by that time. Patients with sustained positive serology are at a minimum in both groups.

Congenital syphilis of 3-24 months duration is somewhat analogous to acquired syphilis during the successive stages leading into latency[5, 6]. Clinical relapses occur infrequently and the tendency to serofastness in the face of adequate treatment increases. The attainment of seronegativity is more closely related to the duration of infection than to the degree of positivity at time of treatment or to the amount of penicillin administered. However, regardless of the amount of treatment or the duration of infection when treatment is administered, seroresistance occurs much more frequently following treatment for congenital than for early (primary, secondary, and early latent) acquired syphilitic infection.

One-half of the treatment failures, one treatment death and four patients who needed additional therapy, occurred among children less than 3 months of age at time of treatment. This may be due to the fact that the less-than-3-months age-group is comprised solely of patients with definite clinical symptoms, which would indicate severe syphilitic involvement and increase the probability of clinical or serologic failure. Furthermore, all of the failures in this age-group received 675,000 units or less of penicillin. Aside from the consideration that this limited quantity of penicillin might not have been adequate for successful outcome, there is the added possibility that the small dosage was dictated by the poor physical condition of these infants which in itself would decrease the chances of the success of treatment.

From the material presented in Table 6 it would appear that the optimum dosage of penicillin in the treatment of congenital syphilis in children 2 years of age or younger probably is 321,000 units per kilogram of body weight. On the basis of studies em-

ploying predominantly aqueous solutions of sodium or potassium penicillin G, the following dosage levels for these rapidly excreted preparations are suggested for the various age-intervals*:

Under 3 months	1,200,000 units
6 months	2,400,000 units
1 year	3,000,000 units
18 months	3,600,000 units
2 years	Adult level (4,000,000-4,800,000 units)

From these data, extrapolation can be made in terms of the delayed-action types of penicillin as demonstrated by therapy evaluation studies in primary, secondary and asymptomatic central nervous system syphilis. The high rate of negativity obtained with POB (Table 6) is a significant finding since it indicates that the delayed-absorption preparations of penicillin are effective in the treatment of congenital syphilis. The use of these types of penicillin in the treatment of children is particularly desirable because it obviates the necessity of administering multiple injections.

SUMMARY

1. Penicillin therapy for congenital syphilis in children 2 years of age or younger is evaluated in terms of serologic status and clinical or serologic failure during a 2-year posttreatment period. The limited follow-up period precludes the determination of the effectiveness of penicillin in preventing the occurrence of late clinical manifestations of congenital syphilis.

2. On the basis of patients followed 18-21 months after treatment, a significantly higher rate of seronegativity was attained in those children treated during the first year of life, regardless of the total amount of penicillin administered.

3. All clinical or serologic failures occurring among those treated during the first 3 months of life were among those receiving less than 800,000 units of penicillin. All other children in this age-interval were seronegative at 18-21 months after treatment.

* Estimates based on age-weight tables: Figures to 1 year from Jeans and Mariott, Infant Nutrition, 4th ed., 1947; figures for second year from Jackson and Kelly, Pediat. J., Sept. 1945.

4. The pattern of serologic behavior in congenital syphilis following treatment during the first 2 years of life is analogous to that in acquired syphilis through the early stages of infection and latency.

5. On the basis of this and other studies employing aqueous solutions of sodium or potassium penicillin G, the minimum dosage for the successful treatment of early congenital syphilis is 321,000 units of aqueous penicillin G per kilogram of body weight, or, roughly, a total of 1,200,000 units to adult dosage level (4,000,000-4,800,000 units) according to the age of the child under treatment. Favorable results among the few patients treated with POB indicate that equivalent dosages of the delayed-absorption types of penicillin are effective in the treatment of early congenital syphilis.

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RESULTS OF ANTIBIOTIC TREATMENT OF PFEIFFER MENINGITIS. (Ugeskr. laeger, 116:1069-1072, July 22, 1954). The diagnosis of Pfeiffer meningitis was confirmed bacteriologically in 31 patients (30 children, mostly under 3 years of age, and one adult) treated in Odense Hospital from 1932 to 1954. Of the 10 treated before the use of streptomycin therapy only one patient survived, while 20 of the 21 treated with streptomycin recovered. Supplementary treatment of meningitis due to Pfeiffer's bacillus with chloramphenicol may result in even greater improvement in the prognosis, and chloramphenicol is now given regularly in addition to streptomycin to all patients with the disease. The procedure is to give sulfonamide, penicillin, streptomycin, and chloramphenicol in the usual dose as soon as purulent meningitis is diagnosed, and when Pfeiffer meningitis is established bacteriologically treatment is continued with only streptomycin and chloramphenicol until the temperature has been normal for four or five days.—*J.A.M.A.*

LEUKEMIA ONE HUNDRED YEARS AGO*

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Two publications, both dealing with the foremost problem in hematology, leukemia, induced me to write these historical remarks —one paper, published in 1854, the other one in 1954, just 100 years later. They illustrate the tremendous increase of knowledge and the changed attitude of the medical profession, from pessimistic therapeutic nihilism to reasonable optimism and hope for a cure of the disease.

In 1854 the first volume of Virchow's Handbuch der Speziellen Pathologie und Therapie was published. Here, for the first time, an attempt was made to describe the clinical picture of the newly discovered pathological entity, "white blood." The author, J. Vogel, states with resignation that nothing is known about the etiology of leukemia and that all therapeutic efforts are ineffective—not much change was made during the following decades. Many theories were offered and discarded; patients suffering from leukemia died, and now, just 100 years later, an optimistic outlook appears. Two sentences from Dameshek's article, "The Outlook for the Eventual Control of Leukemia" may be cited to illustrate the present concept. "There are indications that leukemia, instead of being a hopeless disease, may eventually be put under control" and "it is possible that at any moment some lucky or prepared person will come upon a cure for the disease."

At the present moment, when hematologic research has attained such a climax that hope arises for an understanding of the cause of leukemia and for an effective treatment, it may be interesting to transport the mind into ages past and note how a century ago physicians struggled to understand the peculiar deadly affection called white blood.

White blood was discovered by Rudolf Virchow in 1845 and recognized as a new affection, a morbid entity, pathologically exactly defined. During the following years a few casuistic contributions were published, mostly reporting post-mortem findings. The first paper describing the clinical picture, "The Symptoma-

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tology of Leukemia," was published in 1854 by Dr. Julius Vogel, Professor of Medicine at the University of Giessen (Germany). He wrote the chapter "Störungen der Blutmischung" (disturbances of the contents of the blood) in the first volume of Virchow's Handbuch. He states: "Besides the real, the red blood corpuscles, there are in the normal blood many others, colorless cells, lymph-corpuscles; their significance and function is completely in the dark" and continues: "Only the increase of these cells, the leukemia or leukocythemia, is of practical significance." Leukemia is defined as "an extreme increase of the colorless cells and a decrease of the red cells simultaneously."

In order to understand this point of view, we have to remember that, at the middle of the past century, nothing was known about the white blood cells. Methods for staining were not yet discovered. A droplet of unstained blood could be examined microscopically and only quantitative changes in the relation between the red and white blood cells were studied. The generally accepted theory about leukopoiesis is clearly expressed by Virchow in his study "Weisses Blut und Milztumor." He declares: "Blood is a transitory tissue with a fluid intercellular substance, undergoing continuous evolutions; without intermission the blood gets new young tissue elements, cells. Under normal conditions the vast majority of these cells develop to specific bloodcells, the hemoglobin containing red corpuscles. Under pathologic conditions a disturbance causes an inhibition of the transformation to red cells, whereas the formation of young, unspecific cells is stimulated; the latter are the colorless or white blood corpuscles." This is a very interesting theory—leukemia caused by a kind of maturation arrest; bloodcells originated in the spleen remain colorless white cells and do not mature to red cells. It is a fact hard to understand that neither Virchow nor any other pathologist had examined the bone marrow. The discovery of marrow as the blood forming organ and its characteristic changes in leukemia came 25 years later by Neumann, opening a new era in hematologic research. About 1850 it was generally accepted to consider the spleen as the blood forming organ, "the viscus sanguinificans Vesalii." "If it is not the spleen, I wonder where else the white cells might come from," remarked Virchow.

Virchow distinguished two types of leukemia: the splenic form,

with a greatly enlarged spleen and numerous large white cells; the lymphatic type, characterized by local or generalized lymph node swelling and small white cells in the blood.

Vogel's paper is based on 25 observations, 16 males and 9 females. The author, who proudly claims to have been the first in Germany to make the diagnosis leukemia in vivo, describes the affection as a chronic disease with a slow insidious onset and a progressive fatal course. He enumerates the following signs and symptoms: fatigue, general malaise, diarrhoea, sometimes coughing and dyspnoea, fever, very often nose bleeding and other hemorrhagic manifestations; intensive pallor, enlargement of spleen and liver, and in some cases of the lymphatics are the outstanding phenomena. He gives the following statistical data: in 19 cases observed, the spleen was enlarged 16 times, the liver 13 times and the lymph nodes 11 times. Vogel stresses the point, that the diagnosis, leukemia, is based on the excessive increase of the white cells; their amount may be 1/6, 1/4, 1/2 of the red cells. This can be demonstrated:

1. Microscopic examination of the blood will show the changed relation between white and red blood corpuscles.
2. Sedimentation of a sample of defibrinated blood; the tip of the column shows a whitish gray color.
3. The crusta phlogistica as well as
4. The coagulated blood in the vessels of the body exhibit a grayish white decoloration.

He also mentions the increase of uric acid in the urine—and that is all that laboratory methods could contribute.

Vogel concludes his article with the depressing statement that the prognosis is always fatal, that no treatment is known which might influence the progressive course of leukemia. Vogel, in full agreement with the contemporary physicians, believed that the excessive amount of the white cells in the blood stream is the disease leukemia.

Then he starts speculating: "Is the blood alteration a consequence of the engorgement of spleen and lymph nodes or is it the other way? Is the excessive amount of white cells the primary lesion which causes the enlargement of the spleen and lymphatics? Or could it be that both conditions are originated by an identical, unknown agent? And how does the abnormal amount of white

cells act on the various organs of the body?" Dr. Vogel asks these questions and does not even try to offer an answer.

It is interesting to note that similar considerations were expressed years before by the famous French physician Velpeau (1827): "Does the condition of spleen and liver cause the decomposition of the blood, or, could it be that the abnormal fluid produces the enlargement of these organs?"

Virchow has introduced the "white blood" as a new morbid entity into the pathology. This was an important achievement showing Virchow's genius. One has to remember the obstacles he had to overcome. The train of thought by the contemporary physicians was: the white cells in the blood are "pus-cells," cells always present in suppuration. A considerable amount of those elements indicates an inflammation of the blood, "hemitis," or were taken as a sign of pyaemia. Virchow had to fight against the conservatism of the contemporary physicians, who did not like to accept a new idea. In his first publication he stressed the point, that "white blood" is a new morbid entity, *toto caelo* different from pyaemia, and for more than five years he had to fight for the introduction of the new truth and to reject, time and again, the concept of the pyaemic character of the blood alteration. And when finally his conception was generally accepted, he had to fight against the same pathologists who now claimed priority.

Virchow was the first, who introduced the term "leukocytosis." He states: "A more limited increase of the white cells can be found in a great number of physiologic and pathologic conditions; this can be named leukocytosis." He mentions the leukocytosis after hemorrhages, accompanying infectious diseases, etc. He wrote prophetically: "I prophesy for these leukocytes an important place in pathology." This was said 100 years ago.

At this time leukemia was looked upon as a peculiar, incomprehensible affection, a chronic ailment progressive to an unavoidable deadly end; no therapeutic procedures were known, only vague hypotheses were offered to explain the etiology.

And now, in 1954, the aspect has favorably changed. The frenetic efforts during the past decade, immunbiologic cell studies, animal experiments and the discovery of effective chemicals have demonstrated that leukemia is no longer an absolutely hopeless disease. The most important achievement of modern hematology

is the proof that *leukemia is a reversible* affection. With modern chemicals, remissions are achieved with complete clinical and hematological recovery. The periods of well-being become longer by improved therapeutic technique from weeks to months and years, and there is reasonable hope that a new and improved chemical may prolong the remission for lifetime, and in this way may keep the blood dyscrasia, leukemia, under control.

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ACUTE HYPERVITAMINOSIS A. (Maandschr. kindergeneesk., 22:271-279, Aug. 1954). Hooft presents the histories of four infants ranging in age from 4 to 5½ months, in whom protrusion of the fontanelle was observed after the administration of large doses of vitamin A, usually in combination with vitamin D. Three of the children received 100,000 units of vitamin A, and one received 200,000 units of vitamin A. The doses of vitamin D ranged from 100,000 to 600,000 units. In three of the children the vitamin content of the blood was ascertained. It was normal in two and elevated in one of the children. In this last case the blood was examined 24 hours after the administration of vitamin, in the other two 48 hours later. The first report about the protrusion of the fontanelle after the administration of vitamin A appeared in 1951. In that report it was demonstrated that vitamin A rather than vitamin D was responsible. Other manifestations of increased intracranial pressure may be present, but they are not as constant as the protrusion of the fontanelle, which French authors have referred to as "bombement en chapeau de clown." Hooft believes that the temporary hypervitaminemia, the age of the patient, the nature of the preparation used, the absorption in the gastrointestinal tract, and the reactivity of the liver and the reticuloendothelial system are important factors in the causation of acute hypervitaminosis A.—*J.A.M.A.*

DEPARTMENT OF ABSTRACTS

CHAMBERS, W. R.: ANOREXIA, IRRITABILITY AND CONVULSIONS: EVIDENCES OF SUBDURAL HEMATOMA IN INFANCY. (American Journal Medical Sciences, *228*: 540, Nov. 1954).

Subdural hematoma in infants is a condition in which the history and signs may be misleading and confusing. Its incidence is therefore probably higher than is generally suspected. The consequences of inadequate treatment are so serious that all physicians should be on the lookout for such cases. Thorough treatment by craniotomy with removal of the membranes has been generally satisfactory and the mortality rate has been low. Five representative cases out of the author's experience have been presented.

AUTHOR'S SUMMARY.

FINKE, W.: COMBINED ANTIBIOTIC-CORTISONE THERAPY IN INFECTIOUS ASTHMA. THE RATIONALE OF ITS EARLY APPLICATION. (New York State Journal Medicine, 54: 2685, Oct. 1, 1954).

Combined antibiotic-cortisone therapy seems rational in infectious asthma because such treatment combats two outstanding causes of the disease, bronchial infection and bronchial inflammation. Thirty-eight children and 40 adults with infectious asthma in various stages were given antibiotics for an average of 2¼ years and additional cortisone for an average of 3½ months. The results of treatment were excellent or good in 80 per cent of the whole group. The improvement often approached cure in early cases, as demonstrated by decreasing respiratory sickness and by increasing vital capacity and weight in children. It is concluded that antibiotics and cortisone should be utilized not only to relieve and rehabilitate advanced cases of asthma with irreversible pulmonary pathology but even more as an early causal therapy to prevent a common and costly disease.

AUTHOR'S SUMMARY.

FISHER, O. D. AND FORSYTHE, W. I.: MICTURATING CYSTOURETHROGRAPHY IN THE INVESTIGATION OF ENURESIS. (Archives Disease in Childhood, *29*: 460, Oct. 1954).

One hundred and thirty-five children with persistent enuresis

were investigated by micturating cysto-urethrography. In 43 boys and 31 girls the enuresis was considered to be of functional origin. In 41 boys and 20 girls this procedure revealed abnormalities of the urinary tract. These included valves of the posterior urethra (25), neurogenic disorders of the bladder (4), meatal stenosis (2), ureteral reflux (2), stenosis of the membranous uretha (1) and congenital hypertrophy of the vesical neck (1). The symptoms and signs of the organic disorders did not distinguish them from the functional enuretics. The radiological technique is simple enough to be undertaken in any radiographic department and it causes no more disturbance to the child than catheterization. It is our opinion that micturating cysto-urethrography is an essential investigation for the detection of organic disorders of the urinary tract in children with persistent enuresis.

AUTHORS' SUMMARY.

SNYDER, W. H., JR. AND CHAFFIN, L.: SURGICAL MANAGEMENT OF UNDESCENDED TESTES. (Journal American Medical Association, 157:129, Jan. 8, 1955).

From an examination of evidence in the literature and from a study of 363 patients operated on, we concluded that all patients with undescended testes, who have a clinical hernia, should be operated on by the time they reach the age of five. Those patients, in whom a testis can be palpated in the inguinal region but cannot be pushed into the neck of the scrotum, should also be operated on by the time they reach the age of five, however, in those patients with bilateral undescended testes, in whom there is neither a clinical hernia nor a palpable testis, endocrine therapy should be administered and early surgery should be performed on one side.

AUTHORS' SUMMARY.

HODGSON, J. R. AND KENNEDY, R. L. J.: Bleeding Lesions of the Gastrointestinal Tract in Infants and Children. (Radiology, 63:535, Oct. 1954).

The most common cause of bleeding from the gastrointestinal tract in 246 infants and children seen at the Mayo Clinic were chronic ulcerative colitis, polyps of the colon, Meckel's diverticulum and intussusception. The source of the bleeding was unde-

termined in 19 cases. In this series the most common cause of gastrointestinal bleeding from birth through 1 year of age was intussusception; from 2 through 6 years of age, polyps of the colon; from 7 through 15 years of age, chronic ulcerative colitis. Some of the problems of roentgenologic diagnosis of these and other causes of gastrointestinal bleeding in infants and children are discussed.

AUTHORS' SUMMARY.

MILLER, F. J. W.: Recognition of Tuberculosis in Children under 2 Years. (British Medical Journal, 4892:846, Oct. 9, 1954).

In this paper we have dealt with the recognition of tuberculosis in young children, arguing that diagnosis by clinical signs is outmoded and that early recognition requires above all suspicion, followed by tuberculin testing, radiology, or other investigation. One-third of the 150 ill children presented with illnesses caused by lung primary lesions or miliary tuberculosis; one-third with tuberculous meningitis; one-fifth with superficial lymphadenitis. Bone lesions are relatively uncommon and sensitivity phenomena quite uncommon at this age. The techniques and relative roles of the tuberculin jelly test and Mantoux are considered.

AUTHORS' SUMMARY.

MCMATH, W. F. T.: Measles Meningoencephalomyelitis. (British Medical Journal, 4891:789, Oct. 2, 1954).

Ten cases of measles meningoencephalomyelitis, 3 occurring in the pre-eruptive stage, were encountered in a localized area of North West London between Sept. 1951 and June 1953. The average age of the patients was 5 years, the limits being 20 months and 23 years. No correlation was observed between the incidence and severity of the disease and the character of the primary disease. Of the 3 pre-eruptive cases, one was fatal. This early appearance, and in fact the lack of a fixed time-relationship to the rash, seems to be against the allergic hypothesis of the etiology; moreover, there was no eosinophilia. Coma and severe paralysis are unfavorable features, especially if prolonged, but complete recovery may follow. Predominantly meningitic forms tend to show pronounced fluid changes and encephalitic forms show slight changes, but a high pleocytosis is not incompatible with severe

attacks. Treatment is symptomatic and anticipatory, involving the use of oxygen and chemotherapy for severe forms. Immune sera and concentrated plasma, given intravenously, may turn the scale in selected cases, especially when cerebral edema is a prominent feature. The failure of ACTH to influence the course of the disease (*2* cases) supports the virus rather than the allergic hypothesis of etiology; but the drug was given rather late and in very severe cases. The case mortality, 10 per cent, equals the lowest recorded in a comparable series. While physical residua were negligible or absent among the 9 recoveries, 2 patients, both 3½ years old, showed undoubted mental and behavior deterioration—in one so severe as to require institutional care.

AUTHORS' SUMMARY.

HOWELLS, G.; PALMER, P. E. S. AND ST. JOHN-BROOKS, W. H.: Six Cases of Infantile Scurvy. (British Medical Journal, 4897:1143, Nov. 13, 1954).

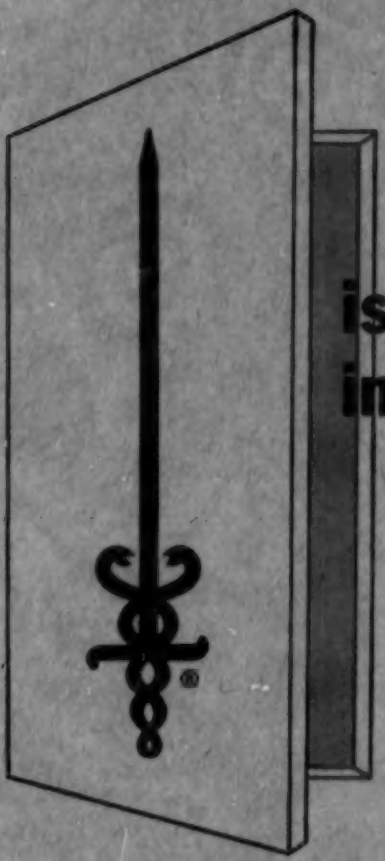
Six cases of infantile scurvy are recorded. Limb tenderness was the most constant positive feature; all the patients were well-nourished and had been artificially fed; all responded to ascorbic acid therapy. The place of radiology in the diagnosis is emphasized. The occurence of these cases provides an important reminder that infantile scurvy—an easily diagnosed and treated disease—still exists, despite modern welfare facilities.

AUTHORS' SUMMARY.

DURIO, A. AND DE STEFANIS, B.: The Use of the Wood's Lamp in the Early Diagnosis of Diphtheria. (Aggiornamento Pediatrico, 5:537, Aug., 1954).

The authors report on their observations of 44 cases of pharyngitis and tonsillitis with the use of the Wood's lamp. The cases ranged in age from 1 year to 67 years. They found that the use of the lamp was helpful in differentiating diphtheritic and non-diphtheritic tonsillitis. In all the cases that were bacteriologically proven to be diphtheritic, a characteristic violet fluorescence of the pharynx was noted. The use of the lamp will help make earlier diagnosis since the color change is noted before the culture growth.

MICHAEL A. BRESCIA, M.D.



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